of ice, bloodletting, not as a routine, but for distinct indications, and serum-therapy, all appear to have been attended with the same general result—a mortality between 50 and 60 per cent.

PERIPHERAL OBLITERATING ARTERITIS AS A CAUSE OF TRIPLEGIA FOLLOWING HEMIPLEGIA, AND OF PARAPLEGIA.

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We purpose to describe a not very infrequent but much neglected form of palsy caused by obliterating arteritis in the extremities affected. When it affects the arteries of the legs in a hemiplegic the result is a triplegia which may be thought to have been caused by cerebrospinal disease if the possibility of local vascular disease and the disability resulting therefrom are not thought of.

During the acute stage of a sudden hemiplegia caused by cerebral hemorrhage or thrombosis, but never in the slowly oncoming palsy from cerebral tumor, there is, on account of the bilateral control of movements in the cerebral cortex, a temporary lessening of power of the arm and leg on the same side as the lesion. It is also well known that in the chronic stage of hemiplegia the deep reflexes on the non-paralyzed side are often permanently increased and that even true ankle clonus may be present. This temporary partial palsy and permanent exaggeration of the deep reflexes arise without any disease of the brain and cord except that which caused the primary palsy. Occasionally, however, the disturbance of function of the leg, which on theoretic grounds should not be affected, is far greater than this, and it becomes as powerless as the other, both thighs being flexed on the abdomen and the calves on the thighs, the final result being a triplegia producing a clinical picture superficially resembling that seen when diffuse myelitis has occurred in a hemiplegic. The condition must not be mistaken for the triplegia which sometimes occurs in syphilis as the result of multiple cerebral and spinal lesions, nor for double hemiplegia or hemiplegia plus monoplegia resulting from bilateral cerebral lesions. It slowly follows a single cerebral apoplexy and is caused, as is shown by post-mortem examination, not by involvement of the spinal cord, but by disease of the arteries of the legs themselves.

The condition does not seem to be very common when the frequency of hemiplegia is considered. It never follows embolic hemiplegia in young people, but occurs only in the aged whose arteries are diseased throughout the entire body.

The history of the case we report is as follows:

W. S., a white man, aged seventy-eight years, was admitted to the Philadelphia Hospital November 16, 1904, with right-sided hemiplegia. His past history is of no interest except that he had had four apoplectic strokes. The last, and the one for which he desired admission to the hospital, occurred about six months before. While standing he suddenly fell unconscious and when consciousness returned was found to be palsied on the right side of the body and speechless. He did not improve in speech or motor power, was weak mentally, and lost control of the bladder and rectum.

Examination. A greatly emaciated old man with spastic paralysis of the right arm and leg. He was completely aphasic, neither speaking nor understanding speech. He made no effort to express thought by pantomime. The reflexes were noted to be increased in each leg, but nothing was said in the notes of this examination as to any loss of power in the left leg. The right pupil was a little larger than the left and both reacted sluggishly to light and convergence. All the peripheral palpable arteries were much thickened. He was bedridden.

When we saw him in January, 1905, both thighs were rigidly flexed at a right angle to the abdomen, and the calves were flexed upon the thighs. The legs were strongly adducted so that the knees could be separated only with difficulty. There was no power of voluntary movement in either leg. The muscular rigidity was so great the legs could not be straightened. The rigidity was not caused by active muscular spasm. The right arm was also completely palsied and contractured in flexion at the elbow and wrist. The knee jerks could not be obtained, but this may have been on account of the extreme muscular rigidity. The muscle jerks were absent. The plantar jerk also was absent. There was no palsy of the left arm. He did not speak at all, paid no attention to anything said to him, and gave no evidence of intelligence. He would swallow food put in his mouth. Sensibility to touch and pain could not be determined because of his mental state. He died on January 15, 1905.

The necropsy revealed chronic myocarditis with disease of the coronary arteries, marked general arterial sclerosis, chronic interstitial nephritis, and areas of cerebral softening. On gross examination of the brain there was found an extensive area of softening involving a large part of the left hemisphere. The only parts that escaped destruction superficially were the first and second frontal convolutions and the anterior portion of the third, the apex of the ascending frontal and ascending parietal, the superior parietal, the third or inferior occipital, the lower part of the second and third temporal, and a small convolution situated in the centre of the softened area in about the position of the lowest part of the ascending parietal, which must have obtained its blood supply from a vessel coming from the interior of the brain at that point. The area of softening surrounded this spot and it is hardly possible that any fibres from it escaped destruction. The mesial surface of the left hemisphere of the cerebrum was normal. On the right hemisphere there was an area of softening involving the calcarine fissure practically in its whole length (about 3 cm.) and about 1 cm. in depth. It was entirely on the mesial surface, the lateral surface of the right hemisphere appearing normal. On the basal aspect the convolutions were normal. The arteries at the base of the brain were intensely sclerotic. On section the area of degeneration in the left hemisphere was seen to extend into and involve the anterior and part of the posterior limb of the internal capsule so that fibres from any part of the left cortex except the occipital lobe would be interrupted in some part of their course. The internal capsule on the right side appeared normal. On microscopic examination the right internal capsule was found normal by the Weigert hæmatoxylin and hæmalaun acid-fuchsin methods. The left internal capsule was largely involved in the lesion of the left hemisphere. There were a few normal fibres in the posterior part of the posterior limb, but most of them appeared degenerated by the Weigert method. The right cerebral peduncle was normal. No degeneration was seen in it by the Weigert method. Sections from the left cerebral peduncle showed the whole crusta distinctly smaller than the right and the nerve fibres of the pyramidal tract completely degenerated by the Weigert method. There was an area in the internal quarter of the crusta which was only partially degenerated. Turck's column was degenerated, showing that fibres from the second and third temporal convolutions must have been interrupted in some part of their course. Sections from the pons showed complete degeneration of the pyramidal tracts on the left side by the Weigert hæmatoxylin method. On the right side they were normal. In sections from the medulla the left pyramid was seen to be entirely degenerated by the Weigert method. The right pyramid and other parts of the medulla were normal by the Weigert and the hæmalaun acid-fuchsin method. Sections from the cervical region showed degeneration, by the Weigert method, in the left direct pyramidal tract and the right crossed pyramidal tract. The small intramedullary bloodvessels appeared very prominent in sections stained by the hæmalaun and acid-fuchsin stain because their walls were thickened. Sections from the midthoracic region were similar to those from the cervical region, the degeneration being confined to the left direct and right crossed pyramidal tracts. Sections from the second lumbar showed no other changes than above described. In the third lumbar the degeneration in the left direct pyramidal tract by the Weigert method was very slight, but distinct. In the right crossed pyramidal tract it was much the same as in the second lumbar. In the fifth lumbar there was distinct degeneration in the crossed pyramidal tract. The bloodvessels of the pia and also in the substance of the spinal cord and medulla showed marked arterial sclerotic changes, but no signs of inflammation or round-cell infiltration. The cells of the anterior horn of the spinal cord in the cervical region were normal by the thionin stain, but they were considerably pigmented and one or two showed slight granular changes in the chromaphilic elements. The cells in the anterior horn of the lumbar region, second, third, and fifth segments, were the same as in the cervical region. The optic chiasm and the optic nerves were normal by the hæmalaun acid-fuchsin and Weigert hæmatoxylin stains. In short, there was nothing in the brain or cord to explain the condition of the left leg. The cord showed only the degeneration proper to be found in the right-sided hemiplegia. The conditions under which the necropsy was made prevented an examination of the bloodvessels, nerves, and muscles of the legs; so that it would be hazardous to draw any conclusions from this case alone. Fortunately we were enabled soon after to examine a case of paraplegia surely caused by arterial disease in the legs, and in which spinal-cord disease competent to produce the condition was absent. Since the arterial condition was the same in both cases, and since there was no disease of the brain or cord competent to explain the symptoms, and since pseudopalsy is well known to be caused by peripheral arterial disease, we think it explains the first as it surely does the second case.

The history of the second case is as follows:

G., a white man, aged sixty-eight years. He was senile and could give no accurate description of his illness on account of poor memory, which he himself realized. He lay in bed with the legs flexed on the thighs and the thighs on the abdomen. The muscular rigidity was very marked. He could flex and extend the thighs and calves a little and could lift the foot a short distance from the bed, but of course could not walk. Attempts at passive movements of either leg immediately increased the rigidity, which, however, could be overcome. There was a very slight occasional knee jerk on the right side, but none on the left. The muscle jerks were absent. He had incontinence of urine and feces. Some weeks before death sensibility was normal on the legs, but whether they became anæsthetic before death we do not know. There was no deformity of the spine nor pain on pressure over it. There was slight muscular wasting in both legs. Later he developed gangrene of the foot and died February 1, 1905.

NECROPSY REPORT. The immediate cause of death was the rupture of an intercostal artery. There were also general arterial sclerosis of high grade, general senile atrophic changes in the abdominal viscera, cardiac hypertrophy, valvular sclerosis, and coronary atheroma.

The brain, spinal cord, and pieces of the posterior tibial artery, nerve, and muscle from each side were removed and preserved in Müller's fluid. The brain was well developed and there was no atrophy or areas of sclerosis. The convolutions were somewhat flattened over both hemispheres. The dura was normal. The pia was slightly thickened, but not adherent. The arteries of the brain, especially at the base, were very sclerotic. The lateral ventricles were quite dilated. The distance between the posterior horn of the lateral ventricles and the posterior pole of the brain was only one cm.; from the lateral wall of the posterior horn to the parietal surface was 2.5 cm., and from the anterior horn to the anterior pole was 3 cm. The ependymal lining of the ventricle, especially of the anterior horn, was distinctly roughened. The cortical gray matter was about normal in thickness. The foramen of Monro was distended and the choroid plexus cystic. The basal ganglia and internal capsule were normal. The aqueduct of Sylvius was patulous, the fourth ventricle about normal in size, and serial sections of the medulla showed no obstruction in the central canal below the fourth ventricle. The cerebellum, pons Varolii, medulla oblongata, and spinal cord appeared normal on gross examination.

The cortex of the paracentral lobules on each side was microscopically normal. The pia was slightly thickened. The Betz cells contained a large but (considering the patient's age) not abnormal amount of yellow pigment situated at one pole of the cell. The chromophilic elements generally stained well, a few cells only showing a granular appearance of the Nissl bodies. The bloodvessel walls were thickened, but there was no round-cell infiltration about them in the pia or cortex. Sections from the optic chiasm and from the optic nerves appeared normal when stained with the hæmalaun and acid-fuchsin and by the Wiegert hæmatoxylin methods. Sections from the medulla oblongata stained by the hæmalaun and acid-fuchsin methods apeared normal but for thickening of the walls of the bloodvessels both in the pia and medullary substance. The anterior pyramids were well stained by the Weigert hæmatoxylin method. Sections from the cervical region of the spinal cord stained by the hæmalaun acid-fuchsin method did not show the changes found in arterial sclerosis of the spinal cord—namely, an overgrowth of glia tissue about the periphery and about the bloodvessels of the cord, with a disappearance of nerve fibres. The bloodvessels in the pia and in the substance of the cord were distinctly thickened, but there was no overgrowth of glia about them. The pia was not thickened and there was no round-cell infiltration in it or about the bloodvessels in the cord.

The Weigert hæmatoxylin method showed distinct degeneration in the inner portion of Burdach's columns and in the columns of Goll, especially in the posterior portion. The Weigert method showed no degeneration of the posterior roots and entrance root zones. Sections from the cervical cord stained by the Marchi method showed recent degeneration in the entrance root zones and in the columns of Burdach. It was very slight in the columns of Goll. Fibres entering the cord showed degeneration by the Marchi method, but posterior roots attached to the section were normal. There was no recent degeneration in other parts of the section. Sections from the thoracic and lumbar region stained by the hæmalaun acid-fuchsin stains showed the same conditions present as described in the cervical region. Sections from the lower thoracic region stained by the Weigert method showed degeneration in the posterior columns more marked in Burdach's columns and the anterior portion of the columns of Goll. The entrance root zone did not show degeneration by the Weigert method. The Marchi method showed marked recent degeneration in the columns of Burdach and in the fibres of the posterior roots after passing through the pia. The posterior roots outside the pia showed no degeneration. Sections from the lumbar region showed a diffuse degeneration of the posterior columns by the Weigert hæmatoxylin method, which, however, did not extend as far forward as the commissure or as far back as the pia. The entrance root zone and the anterior and lateral columns were not degenerated. In sections from the lumbar region stained by the Marchi method the recent degeneration was seen beginning where the posterior roots pass through the pia and extending directly to the degenerated part of the posterior column, the middle third, on each side. There was no degeneration in other parts of the section. Posterior lumbar roots, cut separately from the cord, showed no degeneration by the Marchi method. The cells of the anterior horns of the spinal cord in the cervical, thoracic, and lumbar regions were studied with the thionin stain. They contained a very large amount of pigment. Occasionally the pigment occupied the entire cell and the nucleus was not visible. The chromaphilic substance was usually well stained. In one or two cells (from twenty-four sections examined) there was chromatolysis with displacement of the nucleus to the periphery. Considering the age of the patient, the anterior horn cells may be regarded as normal. The posterior tibial nerve from the right side was almost completely degenerated. The sheath of the nerve was very thick and there was great overgrowth of connective tissue between the bundles of fibres. The nerve fibres themselves in many of the bundles had been replaced by fibrous tissue. But few normal axis cylinders were seen. There appeared to be no inflammatory process or round-cell infiltration. Sections of the left posterior tibial nerve had the same appearance as the right. The right tibial artery showed such marked thickening of the wall that its lumen was almost closed. The left posterior tibial artery was closed by an organized thrombus. Sections from the right and left posterior tibial muscles showed great atrophy. The muscle fibres had a rounded appearance and were separated by an overgrowth of fibrous tissue. Small bloodvessels seen in the sections showed almost complete closure by the thickening of their walls. No nerve fibres were seen in the sections of muscles stained by the Weigert method. The intramuscular nerve filaments had probably degenerated completely. To sum up, there was moderate dilatation of the lateral ventricles, some degeneration of the posterior columns of the cord, great general arterial sclerosis so marked in the right posterior tibial artery as almost to occlude it, while the left was completely closed by an organized thrombus, muscular atrophy, and degeneration of the peripheral nerves. The changes in the spinal cord were not sufficient nor of a nature to have caused paralysis in the legs. The changes in the muscles and nerves surely resulted from the vascular disease. The case, then, was one of senile paraplegia caused by obliterating arteritis. This condition is not very infrequent. It may come on slowly with dull leg pains, paræsthesias, and gradual loss of muscular power, or if thrombi form the onset may be sudden and the result gangrene. In many cases in the earlier stages intermittent lameness occurs.

SOME PHASES OF THE NEUROTIC HEART.

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To my mind there are few chapters pertaining to general medicine less well understood practically than the recognition and judicious treatment of nervous disorders of the heart. I would assign several reasons for my belief in this statement, which is the result of numerous continued observations dating back through many years. First, many of our inquiries to-day, especially those we are apt to regard as more accurate and complete, are made in dispensaries and hospitals, particularly the latter. Usually we regard a case as insufficient and unsatisfactory where an autopsy has not completed our record, or an evident and radical cure been established. In instances where the patient has been more or less of a sufferer during many years, and never seems entirely well, we have reasonable doubts, frequently, as to the accuracy of our diagnosis. This depends upon the fact that, seen at different times and under various conditions, the patient varies a great deal as to symp-